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Infection with *Mycobacterium fortuitum* during acupoint embedding therapy

To the Editor: Acupoint embedding therapy (AET), also known as needle embedding therapy, embeds absorbable foreign substances such as polydioxanone sutures in acupoints to achieve their long-term stimulation. AET has been used to treat chronic and painful disorders, particularly in traditional oriental medicine,¹ and is currently also used for aesthetic purposes (eg, reducing facial wrinkles). However, little is known about the adverse effects of this therapy. Here, we report a patient with nontuberculous mycobacteria (NTM) infection induced by AET.

A healthy 44-year-old woman presented with multiple tender erythematous papules and nodules on both cheeks (Fig 1). She had recently undergone 2 courses of AET separated by an interval of 1 month. Needles were used to embed sterile polydioxanone sutures in acupoints of lower aspects of both cheeks and nasolabial folds. After the second course, multiple oozing erythematous papules and tender nodules appeared, the locations of which corresponded with the points at which the sutures had been embedded. The patient did not have a fever or lymphadenopathy, and responded poorly to acupuncture performed by the oriental medicine doctors who performed the AET. The patient underwent a 1-month course of combined minocycline and cefixime therapy at another dermatology clinic. A skin biopsy specimen revealed a dermal abscess comprising lymphocytes, neutrophils, and histiocytes without foreign body granulomas or suture materials (Fig 2). Dermal tissue culture revealed atypical mycobacteria, which were identified as *Mycobacterium fortuitum* by polymerase chain reaction hybridization using the *rpoB* gene. The isolate was susceptible to doxycycline, ciprofloxacin, amikacin, and clarithromycin. The patient responded well to combination therapy with oral doxycycline, ciprofloxacin, and clarithromycin. After 3 months of treatment, the lesions continued to improve and a 6- to 12-month course of combination therapy was recommended.

AET is thought to work by inducing the release of neurochemicals in response to the application of pressure or needles to acupoints.¹ AET is increasingly used for gradual face lifts and skin tightening, especially among oriental medicine doctors in



Fig 1. *Mycobacterium fortuitum* infection associated with acupoint embedding therapy. Multiple tender, erythematous nodules with a linear distribution on the lower aspect of right cheek and around the nasolabial fold.

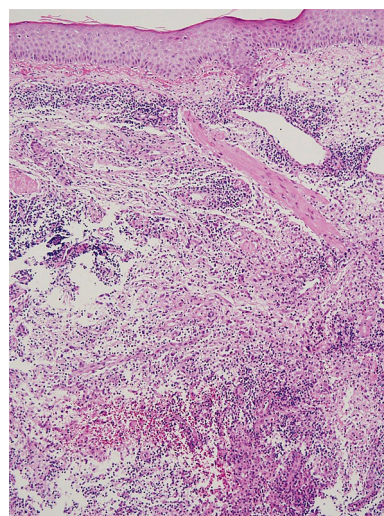


Fig 2. *Mycobacterium fortuitum* infection associated with acupoint embedding therapy. Acute mixed inflammation composed of lymphocytes, neutrophils, and histiocytes involving the entire dermis. (Hematoxylin-eosin stain; original magnification: $\times 100$).

Korea. AET is advertised as a nonsurgical procedure that uses natural materials; therefore, many patients assume it is safer than filler or botulinum toxin treatments. However, AET can cause iatrogenic infections.

As with other strains of NTM, *M fortuitum* infections are generally associated with the use of contaminated solutions and/or equipment during medical procedures. In the case presented here, the source of contamination responsible for the NTM infection could not be identified. However, because disposable needles and threads were used, we suspected that mycobacteria on the patient's skin may have been the source of the infection. The patient was treated with minocycline, which might have a lower benefit/risk ratio than doxycycline.² Until the causative organisms are isolated, empirical antibiotics should be used carefully.

There are reports of NTM infections being contracted after tattooing^{3,4} and acupuncture.⁵ However, to our knowledge, this is the first reported case of *M fortuitum* infection associated with AET. Thus, practitioners should suspect mycobacterial infection in patients presenting with nodules and abscesses in areas that have been treated with AET. Such infections should be treated promptly to minimize further disfigurement.

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Severe radiation dermatitis associated with concomitant vemurafenib therapy in a patient with metastatic melanoma

To the Editor: A 31-year-old woman was diagnosed with a nonulcerated 2.01-mm deep melanoma on her left arm. She underwent wide excision with negative surgical margins and a negative sentinel lymph node biopsy. Seven months later, she started having left hip pain that became progressively worse. A noncontrast

magnetic resonance image revealed a large lesion in her left proximal femur that was subsequently biopsied, confirming the diagnosis of metastatic melanoma. Subcutaneous lesions on her trunk and thighs also developed. Biopsies revealed melanoma with positive BRAF V600E mutation. She was started on vemurafenib, a selective BRAF V600E inhibitor,¹ dosed at 960 mg twice daily. After 3 weeks of vemurafenib, she began receiving localized radiotherapy to treat her femoral lesion. After 1 week of daily radiotherapy, a brisk, raised, erythematous skin reaction restricted to her left anterior and posterior thigh developed. The erythematous regions were confined to the areas where she was receiving radiotherapy. With continuation of radiotherapy for 3 more days, an acute, rapidly worsening, and extremely painful burning sensation developed in her left anterior and posterior thigh. Physical examination revealed blistered and erythematous skin with dry and moist desquamation in the radiotherapy fields on her left anterior and posterior thigh, indicative of a worsening skin reaction consistent with radiation burns. She had received a total radiation dose of 30 Gy in 10 uninterrupted daily 3-Gy fractions as originally planned. With discontinuation of radiotherapy, her radiation dermatitis healed after 1 month.

Two cases of localized radiation dermatitis that resolved with topical corticosteroids and did not require cessation of vemurafenib have been reported in patients who started vemurafenib after completion of radiotherapy.² Another case of radiodermatitis was reported in a patient receiving concomitant vemurafenib and radiotherapy who had been earlier administered 60 Gy of local radiotherapy to the same area that 4.5 years later developed radiodermatitis upon administration of 20 Gy of radiotherapy.³ To our knowledge, our case represents the first reported instance in the English literature of localized radiation dermatitis developing in a patient receiving concomitant vemurafenib and radiotherapy who was previously naive to both treatment modalities. Sustained erythema seen in radiation dermatitis typically manifests 10 to 14 days after dosing, whereas sustained erythema developed in our patient after 7 days of radiotherapy. Moreover, the severity of the radiation dermatitis seen in our patient after 10 days of radiotherapy is not usually seen until after 4 to 5 weeks of radiotherapy at doses of 40 Gy or greater. The radiosensitizing effect of vemurafenib leads to increased cellular damage and impaired DNA repair.⁴ One proposed mechanism for radiosensitization secondary to vemurafenib suggests that vemurafenib activates wild-type BRAF in keratinocytes and leads to radiosensitization as a result.³ However, the exact mechanism remains unclear. Dermatologists should